This Week in The Journal

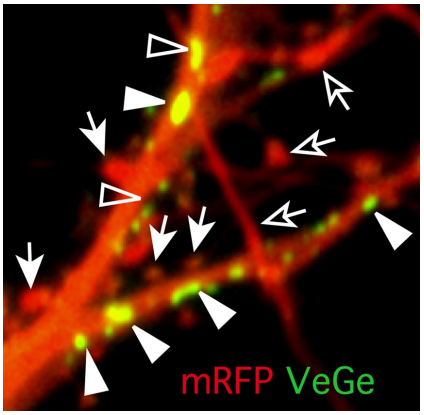
● Cellular/Molecular

Postsynaptic Scaffolds on the Move

Cyril Hanus, Marie-Virginie Ehrensperger, and Antoine Triller

(see pages 4586–4595)

Generally, it's not a good idea to shake or move a ladder sideways with someone on it. However, that seems to be exactly what synaptic scaffolding proteins do. In this week's *Journal*, Hanus et al. monitored the GABA/glycine receptor scaffold gephyrin by tagging it with the yellow fluorescent protein derivative Venus. Cultured neurons were transfected on day 8 and then imaged 1–2 d later. In rat spinal cord neurons, Venus:gephyrin chimeras clustered on postsynaptic dendrites, but time-lapse imaging revealed submicron lateral movements. Gephyrin cluster movements were much faster than global dendritic movements. The authors suggest that fast (<5 seconds) and slower (>10 seconds) components corresponded to shifts within clusters and lateral movement of clusters. Both actin dynamics and microtubules affected the movements, perhaps not surprising given the location of these inhibitory synapses on dendritic shafts. Enhancement of neural activity diminished the scaffold movements.



Dendrites and dendritic spines (mRFP, red) and inhibitory synapses (Venus:gephrin, green) were labeled in a cultured hippocampal neuron. See the article by Hanus et al. for details.

▲ Development/Plasticity/Repair

Migrating with MDGA1

Akihide Takeuchi and Dennis D. M. O'Leary

(see pages 4460–4464)

This week, Takeuchi and O'Leary outline a developmental role for MAM domain glycosylphosphatidylinositol (GPI) anchor1 (MDGA1), a recently cloned Ig cell adhesion molecule. This glycoprotein is anchored to the extracellular membrane by a GPI linkage and is similar in structure to Ig-containing cell adhesion molecules such as L1. Because layer 2/3 mouse neurons expressed MDGA1 during development, but not adulthood, the authors hypothesized that MDGA1 was important for cell migration. They suppressed MDGA1 expression in mouse brains with RNA interference delivered by electroporation *in utero*. Transfections were timed to embryonic day 15.5 so that most transfected cells were destined to become layer 2/3 neurons. Without MDGA1, layer 2/3 neurons were trapped in the intermediate zone or deep in the cortical plate (CP) rather than migrating to the superficial CP that becomes layers 2/3. Overexpression of MDGA1 rescued the migration of layer 2/3 neurons.

■ Behavioral/Systems/Cognitive

The Subdivisions of Visual Working Memory

Harald M. Mohr, Rainer Goebel, and David E. J. Linden

(see pages 4465–4471)

According to the "domain-specific" view, visual working memory tasks are separated in the lateral prefrontal cortex into functional subdivisions: dorsal areas handle the spatial features of a task, and ventral areas take on object and color information. The "process-specific" account, in contrast, argues that the ventral areas maintain information while manipulation occurs in dorsal areas. This week, Mohr et al. attempted to reconcile these two views. Human subjects were shown a colored semicircle and performed either maintenance or manipulation tasks after a delay period. Functional magnetic resonance imaging during the delay showed that cortical activity was segregated based on content. Dorsal premotor activation occurred with maintenance and manipulation of spatial tasks, whereas ventral premotor activation was seen for maintenance and manipulation of color tasks. Manipulation-specific activity involved other areas in a frontal-parietal network, consistent with multiple levels of specialization in frontal cortex.

♦ Neurobiology of Disease

Postseizure Granule Cell Dispersion

Christophe Heinrich, Naoki Nitta, Armin Flubacher, Martin Müller, Alexander Fahrner, Matthias Kirsch, Thomas Freiman, Fumio Suzuki, Antoine Depaulis, Michael Frotscher, and Carola A. Haas

(see pages 4701–4713)

Temporal lobe epilepsy is accompanied by several structural changes in the hippocampus, including widening of the granule cell layer in the dentate gyrus. In this week's *Journal*, Heinrich et al. examined the causes of this granule cell dispersion (GCD) in mice after intrahippocampal injections of kainic acid (KA). This protocol induced repeated hippocampal seizures (nonconvulsive status epilepticus) of up to 24 hours. The seizures were followed by degeneration of ipsilateral CA3, hilar, and CA1 neurons, but apparently complete behavioral recovery. GCD was evident within 7 d, and chronic spontaneous epileptic discharges began 2 weeks after injection. However, the GCD did not appear to result from increased neurogenesis. Reelin-expressing neurons decreased significantly in KA-injected mice, and an injection of a reelin-neutralizing monoclonal antibody caused GCD in control mice. The authors suggest that GCD occurs as a result of either misguided migration or displacement of mature neurons because of localized reelin loss.